1: Microbiol Immunol. 2000;44(4):259-66.

Intranasal sensitization of Japanese cedar pollen by the co-administration of low doses of cholera toxin but not its recombinant B subunit to mice.

Hirai T, Hashiguchi S, Torigoe N, Toda Y, Ito Y, Sugimur K.

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We evaluated the effects of cholera toxin (CT) and the B subunit of cholera toxin (CTB) on the intranasal sensitization of Japanese cedar pollen (JCP) in mice. JCP suspended in phosphate-buffered saline was administered into the nostrils of mice in combination with varying doses of CT or recombinant CTB(r-CTB) once a week for 5 weeks. Antibody responses specific to sugi basic protein (SBP) were monitored by ELISA for seven weeks. The sensitization of JCP alone did not induce IgG1, IgG2b, IgG2a, IgE or IgA. In contrast, sensitization of JCP in combination with CT (JCP/CT) elicited the prominent production of SBP-specific IgG1 and low levels of IgG2b and IgG2a on Day 49. IgE production was detected only in the serum of mice which were treated with JCP/CT, and not under any other protocol. Using spleen cells from these mice, cytokine production was examined by ELISA in culture supernatants after they had been stimulated in vitro with major cedar pollen allergens, Cry j 1, Cry j 2 or SBP. Notable responses were an increase of IFN-gamma as well as IL-4 in JCP/CT-sensitized cells stimulated with Cry j 2, but not in those stimulated with Cry j 1. No significant differences were detected in IL-5 production among the experimental groups. Histopathological examination, however, showed that eosinophil infiltration was evident in the nasal mucosa of the JCP/CT-sensitized mice following challenge with JCP/CT, but weak with BSA/CT or CT alone. Thus, the immunological and histological analyses indicated that the co-administration of a low dose of CT in combination with JCP allows the induction of pollen-allergic states in mice.

PMID: 10832969 [PubMed - indexed for MEDLINE]

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1: J Allergy Clin Immunol. 2003 May;111(5):1122-8. Related Articles, Links

Genetic susceptibility to food allergy is linked to differential TH2-TH1 responses in C3H/HeJ and BALB/c mice.

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BACKGROUND: Although food allergy is a serious health problem in westernized countries, factors influencing the development of food allergy are largely unknown. Appropriate murine models of food allergy would be useful in understanding the mechanisms underlying food allergy in human subjects. OBJECTIVE: We sought to determine the susceptibility of different strains of mice to food hypersensitivity. METHODS: C3H/HeJ and BALB/c mice were sensitized to cow's milk (CM) or peanut by means of intragastric administration, with cholera toxin as a mucosal adjuvant. Mice were then challenged with CM or peanut. Antigen-specific IgE levels, anaphylactic symptoms, plasma histamine levels, and splenocyte cytokine profiles of these 2 strains were compared. RESULTS: CM-specific IgE levels were significantly increased only in the C3H/HeJ strain, 87% of which exhibited systemic anaphylactic reactions accompanied by significantly increased plasma histamine levels in response to challenge. BALB/c mice exhibited no significant CM-specific IgE response, increased plasma histamine levels, or anaphylactic symptoms. After peanut challenge, 100% of peanut-sensitized C3H/HeJ mice exhibited high levels of peanut-specific IgE and anaphylactic symptoms. In contrast, no hypersensitivity reactions were detected in BALB/c mice, despite the presence of significant serum peanut-specific IgE levels. Splenocytes from CM- and peanut-sensitized C3H/HeJ mice exhibited significantly increased IL-4 and IL-10 secretion, whereas splenocytes from BALB/c mice exhibited significantly increased IFN-gamma secretion. CONCLUSION: Induction of food-induced hypersensitivity reactions in mice is strain dependent, with C3H/HeJ mice being susceptible and BALB/c mice being resistant. This strain-dependent susceptibility to food allergy is associated with differential T(H)2-T(H)1 responses after intragastric food allergen sensitization.

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1: Clin Exp Immunol. 1998 Jan;111(1):144-51. Related Articles, Links

Effects of adjuvants on the immune response to allergens in a murine model of allergen inhalation: cholera toxin induces a Th1-like response to Bet v 1, the major birch pollen allergen.

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Based on the fact that type I allergies are frequently elicited by inhalant allergens, we have established a model of aerosol inhalation leading to allergic sensitization in BALB/c mice. Using this model we studied the effects of aluminium hydroxide (Al(OH)3), known to enhance IgE antibody responses, compared with cholera toxin (CT), a potent mucosal adjuvant, on the immune response to birch pollen (BP) and its major allergen Bet v 1. Two groups of BALB/c mice were either systemically immunized with recombinant Bet v 1 in Al(OH)3 and subsequently aerosol exposed to BP allergen, or aerosolized with BP and CT. IgE-mediated skin reactions were only elicited in the mice which had received Bet v 1/Al(OH)3. Allergen-specific serum IgE and IgG1 antibodies dominated in the Al(OH)3 group, IgG2a antibody levels to BP and rBet v 1 were markedly higher in the sera of mice exposed to CT with the allergen. IgA antibodies were only detected in the bronchial lavage of the CT-treated group. Moreover, the latter group displayed consistently higher T cell proliferative responses to BP and interferon-gamma production in vitro. Thus, the systemic immunization with rBet v 1 in Al(OH)3 before inhalation of the BP extract promoted a Th2-like immune response, while CT mixed with the aerosolized BP extract rather induced a Th1-like immune response. In an attempt to reverse these ongoing immune responses we could achieve a shift towards a Th0 response. Immunization with BP extract without adjuvant treatment led to undetectable antibody or cellular immune responses. We conclude from the present study that the induction of an immune response to BP allergen after aerosol inhalation can be directed towards a Th1- or a Th2-like response. Once established, the immune response can be modulated.

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